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EDITORIALS

DISEASE CONTROL

We publish in this issue of the British Veterinary Journal an account of a valuable and important piece of research work, by J. R. Hudson, on yet another tissue vaccine which might be used in the control of rinderpest. The agents now available for this purpose have been evolved following careful and exact research work in the laboratory and in the field, and each has been shown to have a place in different parts of the world. Modern methods of scientific research have been envoked in the investigations and the evolution of the various agents has been the result. The goat, the chick (in the form of the developing embryo), the rabbit, and now the pig have been shown to be useful animals in attenuating the virus to a degree sufficient to render it comparatively safe for general use in certain parts of the world and on certain breeds of cattle. With these weapons in our hands it should now be possible to tackle the problem of rinderpest control on a world-wide basis.

Fortunately, our own country is free from this disease, as it has been for many years following the drastic slaughter policy introduced by our far-seeing veterinary profession at that time. There is no doubt that the slaughter policy as practised then and now is of the utmost value in keeping this country comparatively free from exotic diseases which could and would become established without such drastic action. Those who have to accept responsibility for the control of such diseases and for the prevention of their entry into the country must also, of course, be prepared to advise on methods of control, should the necessity arise. Up to the present, reliance has been placed on a slaughter policy together with abolition and restriction of movement of animals. There

are no indications whatsoever that there is any need to consider any other policy: however, it is important to envisage other methods of control, should the slaughter policy break down, and for this reason we must contrive to take the liveliest interest in all research work which has as its object the devising of satisfactory immunological techniques and procedures and to encourage further studies.

In some other parts of the world a slaughter policy is impossible: therefore, for economic reasons, immunological methods have to be adopted. With a disease like rinderpest, overflowing as it does from one country to another, common action is called for and must be taken if there is to be any real chance of ridding large parts of the world of the infection which plays such an important part in lessening the livestock economy in so many areas. Immunisation with suitable and well-tested agents can be carried out and applied on an ever-increasing international scale. This treatment must form the basis of control measures and may, in the future, lead to the position when the disease is so well controlled that further immunisation may not be necessary. We realise, of course, the difficulties involved in such a project, but it is the duty of those in a position to do so, at least to attempt to bring into operation an international scheme for ridding the world of a disease like rinderpest for the benefit of all nations.

THE OLD YEAR

By the time this issue is published we shall be entering upon another year. We take this opportunity of extending to our readers our best wishes for 1951. The year through which we have just passed has shown that the Veterinary Profession, not only in this country, but in many parts of the world, is fulfilling its purpose and is tackling the problems of animal health in a manner worthy of our training and tradition. We have certainly an important part to play in the economic life, the prosperity and the peace of the world to-day. It is our earnest hope that we will be given continued opportunity to play our part: given the opportunity we have no hesitation in asserting that we will not be found wanting.

XUM

GENERAL ARTICLES

THE UTILISATION OF PIGS FOR THE PRODUCTION OF LAPINISED RINDERPEST VIRUS

By J. R. HUDSON and C. WONGSONGSARN

THE development by Nakamura, *et al.*, in 1938 of a strain of rinderpest virus attenuated for cattle by serial passage in rabbits placed in the hands of those engaged in the control of this disease a new and important weapon. At the F.A.O. Conference on rinderpest in Nairobi, 1948, Cheng and Fischman (1949) gave an excellent summary of the work that has been done on this lapinised virus and described the method adopted in China of preparing and using the virus in the field. They and other workers consider that one of the most important limitations of the method is the necessity for having large numbers of rabbits, animals which appear difficult to rear in quantity in tropical and subtropical countries.

Cheng and Fischman refer briefly to attempts by Cheng (1948) and Kwong (1947) to use calves and goats as virus producers. Material from a calf and from a goat inoculated with rabbit virus was tested on susceptible calves. Material from the calf proved of no value; but blood from the goat protected six out of six calves.

The immunisation of water buffaloes in Thailand necessitates the use of a virus of considerably lower virulence than goat-adapted virus. Fresh lapinised virus prepared from rabbits provokes no thermal reaction in these very susceptible animals. Although no long-term immunity tests have been made, immunity at 14 days is good and it is reasonable to expect a longer duration of immunity than that obtainable with a single dose of inactivated vaccine, a product expensive to prepare and necessitating the slaughter of considerable numbers of an animal much in demand for agricultural purposes.

Field tests in Thailand had shown that, whereas buffaloes and cattle gave no thermal reaction to lapinised virus, some pigs did. This suggested that a high titre of virus might be obtained more readily in the blood and tissues of pigs than in those of the large ruminants. An attempt to establish a line of virus in buffaloes proved unsuccessful. Two buffaloes were inoculated intravenously with 5 c.c. of a 10^{-1} dilution of a suspension of spleen in blood (1 : 20) from two rabbits, the R.845th passage of Nakamura III. There was no rise in temperature; but the buffaloes were bled on the 4th and 5th days after inoculation and blood was inoculated with a minimum delay into two rabbits. These rabbits developed, respectively, a late and a doubtful temperature reaction. They were killed on the 4th day after the second injection and the lesions found were not characteristic. However, a suspension of spleen and lymph glands in blood was prepared and inoculated intravenously into a second pair of buffaloes. These were bled on the 4th and 5th days after injection to inoculate further

rabbits. These rabbits developed no thermal reaction and on slaughter were free from lesions. Both the first pair of buffaloes proved immune on challenge with stock buffalo virus. Of the second pair, one was immune and one susceptible.

Rinderpest in Pigs

Judging from the information available in Hutyra and Marek (1946) opinions on the susceptibility of pigs to rinderpest differ. "In swine subcutaneous injection or feeding with large quantities of virulent blood usually results only in a rise of temperature and is rarely fatal. (Carré and Frimbault, Vrijburg and others). Hutchin found swine almost as susceptible to the virus as zebu cattle, but Theiler, Angeloff and Pirani failed to infect swine. Penning, however, infected wild boars. . . . On the occurrence of spontaneous infection in swine there are different opinions. While there is no mention of such an occurrence in the earlier reports, and it was not observed even in the last outbreak in Europe and most recently in West Africa (Curasson), Boynton maintains that swine can be infected by affected cattle, and Youngberg reports from the Philippine Islands that cattle and swine may be affected at the same time and that the infection was conveyed by pigs to the island of Romblon. The course of a 10^{-1} dilution was injected intravenously into pigs 5 and 6. These pigs also been reported by other authors. According to Turner, fresh outbreaks of rinderpest in the Malay Islands often originate from wild hogs."

Curasson (1932) discusses the matter at length citing evidence from Europe and Africa, that pigs are not infected naturally and also citing authors in the East Indies, Philippines, Cochin-China and Southern India who have seen outbreaks involving pigs. Failures to infect experimentally are reported from Turkey, Bulgaria and Russia and from Africa. Curasson himself tried to infect pigs in W. Africa where natural infection in domestic pigs is quite unknown. Pigs fed on meat from virus-makers did not react. Of four young pigs given large doses of infected blood by the mouth, one showed a thermal reaction. Blood from the reacting pig transmitted infection to a calf but not to another pig. On the other hand, of 15 pigs and piglets inoculated subcutaneously with blood, eight reacted, one showing symptoms analogous to those seen in cattle: the others had either indefinite symptoms or a thermal response only. Cross-bred Yorkshire x local breed pigs were of low susceptibility.

In East Africa, although rinderpest spreads rapidly in, and is very fatal to, warthog, no spontaneous infection of domestic pigs has ever been reported. One of us (J.R.H.) in 1927 saw pigs running on land which was grazed simultaneously by cattle suffering from rinderpest contracted from infected warthog. Temperatures were not taken, but the pigs showed no obvious evidence of infection.

Robertson (1924), in Australia, infected six pigs, presumably of European type. Two were fed with blood, intestines and glands of infected cattle and four were inoculated with citrated blood. There was considerable variation in the thermal response and, whereas some pigs developed diarrhœa and dysentery, others showed no clinical symptoms. All the pigs were killed on the

twelfth day after inoculation to suppress the disease; but from the appearance of the charts, they would probably have survived.

Molinié (1931) describes natural cases of rinderpest in pigs in Indo-China. In spite of the fact that during severe outbreaks in buffaloes, pigs had many opportunities of becoming infected through eating carcasses, deaths in pigs were always uncommon and probably many pigs developed the disease in an inapparent form. Molinié was able to infect pigs by the inoculation of infective blood from natural cases in pigs and buffaloes; but he found that the disease did not spread easily from pig to pig by contact. Vittoz (1950) is also well acquainted with rinderpest in pigs in Indo-China and Crawford (1949) saw outbreaks in pigs of the local breed when in Ceylon. In Thailand, outbreaks of rinderpest in pigs were not uncommon and according to Jones (1949) movement of pigs by train has been blamed as the cause of new outbreaks in cattle and buffaloes.

Brief consideration of the geographical distribution of rinderpest suggests that pigs develop the disease in the eastern part of its range but not in the west. The boundary between the two would seem to be the broad belt occupied by the Moslem populations of Iran and Iraq. In the area where pigs do not develop rinderpest, the pigs may be described as of "European descent." In the east the indigenous pig (Fig. 1) is a small black animal, with small ears, a hollow back and a long, triangular face. It is possible that there is a difference in susceptibility due to breed, but at the moment a difference in host specificity of the wild strains of rinderpest virus cannot be completely excluded.

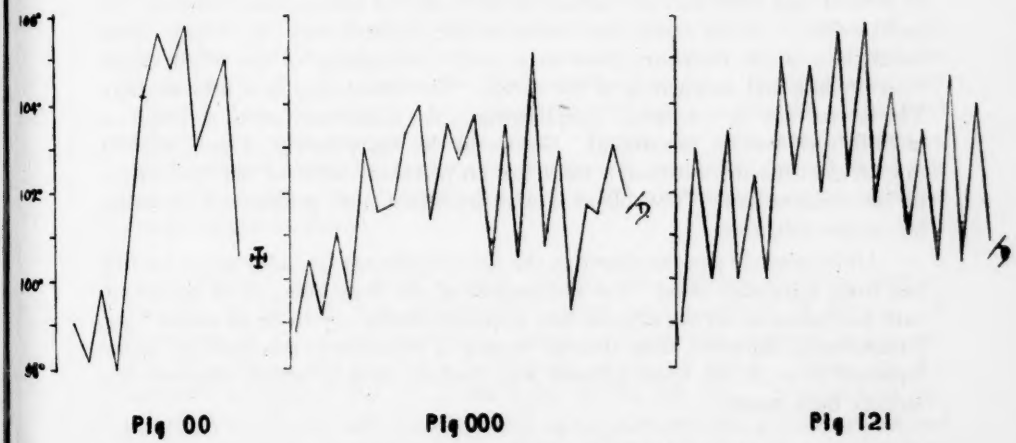

FIG. 2.—Temperature charts of three pigs inoculated with buffalo virus. They illustrate the variation in susceptibility to rinderpest.
[In this and the following figures the temperatures are recorded twice a day. The first record is commonly that of the evening after inoculation; but in the charts of pigs Nos. 000 and 121, the morning temperature on the day of inoculation is given.]
D = *discontinued, apparently healthy.*

Experimentally the pigs obtainable by purchase in Thailand vary considerably in their susceptibility to rinderpest. When inoculated with the stock, Pak Chong, buffalo-virus about 25 per cent develop severe symptoms and die: others give a thermal response only (Fig. 2). Molinié (1931) reported a mortality of 22 per cent in his pigs inoculated with virus of buffalo origin. In severe cases there is diarrhœa and rapid loss in condition. Appetite is decreased and the "vesicles" which are pathognomic of the disease in buffalo may be present around the anus and in the perineal region.

Our findings on the lesions present in pigs that die of rinderpest agree generally with those recorded by Molinié. As in cattle, there is considerable variation in the severity of the lesions present in different organs. The back of the tongue and pharynx may show cyanosis, fine necrotic lesions or severe diphtheresis. In the stomach also, the changes vary from a little hyperæmia of the pyloric region to marked, diffuse congestion and necrosis of the superficial mucosa. The small intestine usually exhibits areas of congestion and the Peyer's patches may appear unduly prominent. There is no necrosis in the Peyer's patches such as is present in rabbits inoculated with the lapinised strain of virus. In the cæcum lesions are usually marked. They consist of areas of congestion, up to three inches in diameter. From the centre, sometimes to the periphery, extends a variable area of necrosis of the mucosa. This necrotic area is covered by a diphtheritic deposit which varies in thickness.

In cases that die late in the course of the disease, these necrotic ulcers may be the sole lesions. They are similar to, but much more extensive than, the ulcers of swine fever. The mesenteric lymphatic glands are usually swollen and congested. The liver has no characteristic lesion. Some congestion may be present and there may be vascular arborescence of the mucosal vessels of the gall-bladder. As in cattle, the lesions in the kidneys vary in degree from hyperæmia of the boundary layer to a marked congestion of the whole organ with swelling and roughening of the cortex. The spleen may be a little swollen. The larynx may be cyanosed. Emphysema of the pulmonary septa, so common in cattle and buffalo, is unusual. Lesions in the heart consist of pale, slightly dry areas in the myocardium: there are no petechiæ either of the epicardium or the endocardium. The blood is dark in colour and produces a prompt, but rather soft, clot.

Unfortunately pigs purchased in the open market are probably never entirely free from European blood. For the purpose of the work about to be described, care was taken to obtain animals that appeared to the eye to be of native type. Undoubtedly, however, these showed as great a variation in susceptibility to the lapinised virus as did those infected with buffalo virus to which reference has already been made.

Attempts to Passage Lapinised Virus in Pigs

When material of rabbit passage R.845 was injected into the buffaloes, 5 c.c. of the same suspension was inoculated intravenously into two pigs, Nos. 1 and 2.

At the time, we considered that one of these pigs (Fig. 3) gave a thermal response and it was killed on the fourth day after inoculation. Defibrinated blood and spleen were collected and a suspension prepared as in the usual technique for the subinoculation of the lapinised strain in rabbits. A 10^{-1} dilution of this suspension was inoculated intravenously in 5 c.c. doses into pigs Nos. 3 and 4 and in 0.25 c.c. doses into rabbits Nos. 3 and 4. The pigs 3

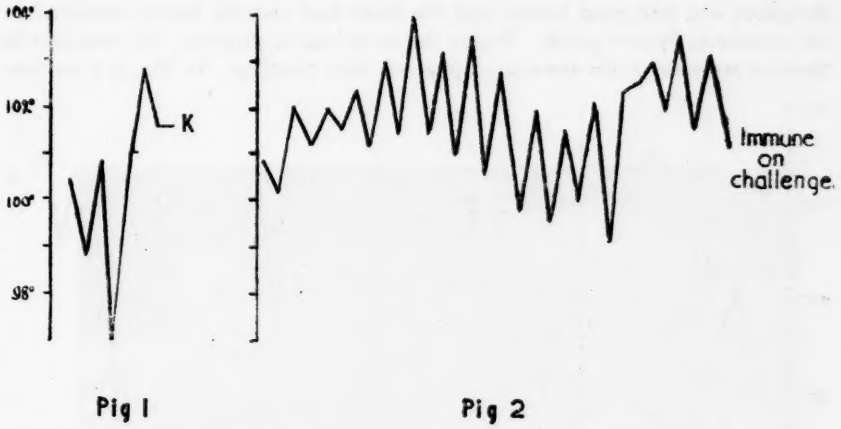

FIG. 3.—Temperature charts of pigs 1 and 2, the first passage of lapinised virus in pigs. K = *killed for material.*

and 4 both appeared to develop a rise in temperature as a result of the inoculation and No. 3 was killed on the 4th day, material being subinoculated to pigs 7 and 8. The strain was then continued in series and was called the "direct" line, the numbers of passages in pigs being numbered. The P6D indicates the sixth passage in this series. During the first three passages, a 10^{-1} dilution of a suspension of 1 part of spleen in 20 of defibrinated blood was used as inoculum; but thereafter 5 c.c. of a 10^{-1} suspension of spleen in saline was given intravenously.

Rabbits 3 and 4 gave a good, slightly late, thermal response and on slaughter on the 4th day, well-marked lesions were found to be present. A suspension of spleen and mesenteric gland in blood was prepared and 5 c.c. of a 10^{-1} dilution was injected intravenously into pigs 5 and 6. These pigs also appeared to respond and No. 5 was killed on the 4th day. Spleen-blood suspension was injected into rabbits which again gave late but good reactions and excellent lesions were present on slaughter. Material from them was inoculated into pigs 12 and 13. In this way an "alternate" line was propagated, the pigs 12 and 13 being described as passage P3A. Usually only one pig of each passage was killed for virus. Most of the others survived and were later available challenge with buffalo-virus.

Direct Passage in Pigs

The direct line was propagated without difficulty 17 times in pigs. Material of the 3rd, 6th, 10th and 17th passages was inoculated subcutaneously into buffaloes which gave no reaction but were protected against challenge with buffalo virus. Material of the 10th passage was inoculated intravenously into two rabbits. Both these rabbits responded with typical thermal reactions. On slaughter, one had good lesions and the other had necrotic lesions restricted to an occasional Peyer's patch. Whilst the work was in progress, the variation in thermal response in the inoculated pigs was very puzzling. In Fig. 4 a number

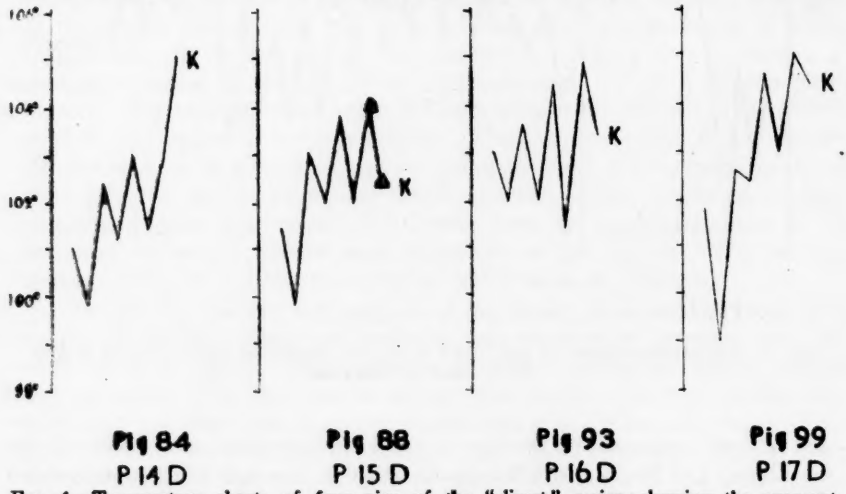

FIG. 4.—Temperature charts of four pigs of the "direct" series, showing the apparent reactions.
The symbol Δ around a reading indicates *slightly off feed.*

of charts are given to illustrate this variation. After the strain was lost, however, apparent reactions continued in some of the pigs inoculated with material now known to have been inert, and comparison of Fig. 4 with Fig. 5 will make it clear that there is no justification for attributing the "reactions" in Fig. 4 to rinderpest. Of the surviving pigs—that is to say, the pigs which were not killed for material—inoculated during the first 17 passages of the direct line, those of passages 7, 11, 13 and 15 died. Diarrhœa was observed in those of passages 8, 13, 14 and 15, and inappetance only in passages 4, 5, 9 and 10. In the pigs which were injected with inert material after the line was lost there was no diarrhœa or loss of appetite. It would appear, therefore, that whereas the thermal reaction could not be attributed to the injection of lapinised virus, the diarrhœa and inappetence were in all probability due to infection with this virus.

Of the pigs that died, pig 37 (P7D) succumbed as the result of toxæmia from septic wounds and pig 60 (P11D) of gastric œdema. These two pigs

FIG. 1.—Pig of the type used in the experiments

(*Article by Hudson and Wongsongsarn, page* 453)

showed no lesions suggestive of rinderpest. Pig 72 (P13D, Fig. 6) died on the 9th day after inoculation, and lesions found *post mortem* suggested that rinderpest was the cause of death. In pig 87 (P15D), death—which occurred during the 8th night—may have been caused by rinderpest, for diphtheritic lesions were present in the pharynx and rectum and there was an acute catarrhal nephritis. No significant lesions were, however, found in the stomach or cæcum. Deaths of pigs 72 and 87 suggested that the virus was either becoming more virulent for pigs or had become contaminated with buffalo virus. The subsequent behaviour of the virus in pig passages P16D and P17D and the results of inoculating P17D to buffalo did not support either of these alternatives.

At the 18th passage the direct line was lost. Neither of the pigs showed any thermal response on the 4th morning after inoculation, the normal day for passaging the virus, and it was decided not to kill until the following day, when pig 103 (Fig. 5) was slaughtered. Possibly, had the other pig been killed, the

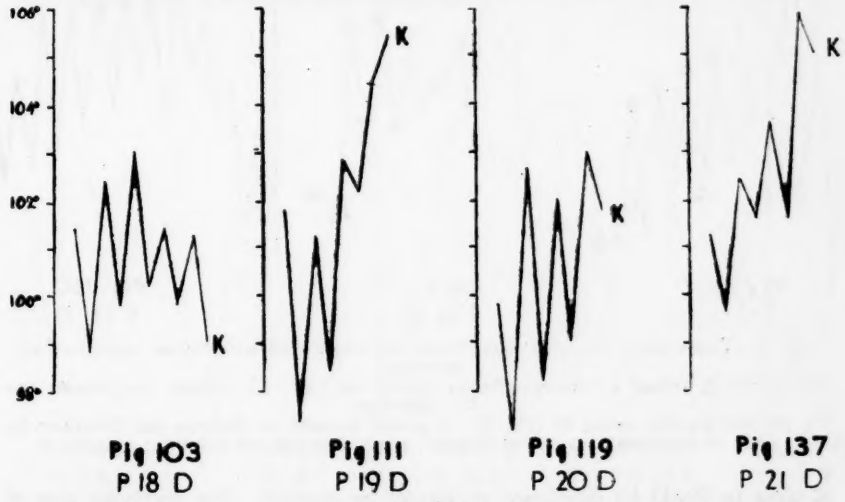

FIG. 5.—Temperature charts of four pigs after the "direct" line was lost and no virus was being passaged.

line could have been kept, for this pig suffered from diarrhœa and inappetence on the 6th, 7th and 8th days. Challenged with buffalo virus on the 23rd day after inoculation, it proved immune.

A suspension of spleen of pig 103 was injected intravenously into further pigs and into rabbits 33 and 34. The temperature curve of pig 111 (Fig. 5) suggested that virus was still present, and spleen suspension from this pig was inoculated in due course into rabbits 39 and 40. Neither rabbit 33 nor 34 reacted. No. 34 was killed on the 4th day and no characteristic lesions were found. Rabbit 33 proved susceptible later, on challenge with rabbit passage R.847 material. Of the rabbits inoculated with P19D, No. 39 gave a doubtful

thermal reaction. Both the rabbits were killed on the 4th day; lesions were absent in No. 39 and of doubtful significance in No. 40. The question of whether the virus had been lost, or whether virus was present but had lost its ability to provoke the typical response in rabbits, then arose. Inoculation of buffaloes with P20D spleen and with P21D spleen, the latter from a pig appearing to give an excellent thermal response, showed that these spleens were unable to protect buffalo against virulent rinderpest. On challenge, the surviving P19D pig gave a slight temperature reaction. Three surviving P20D pigs were available for challenge. All reacted and one pig, No. 124, died of rinderpest.

A further attempt was made to continue the line by inoculating two pigs with dried P17D spleen. Unfortunately, the buffalo used to test for the presence

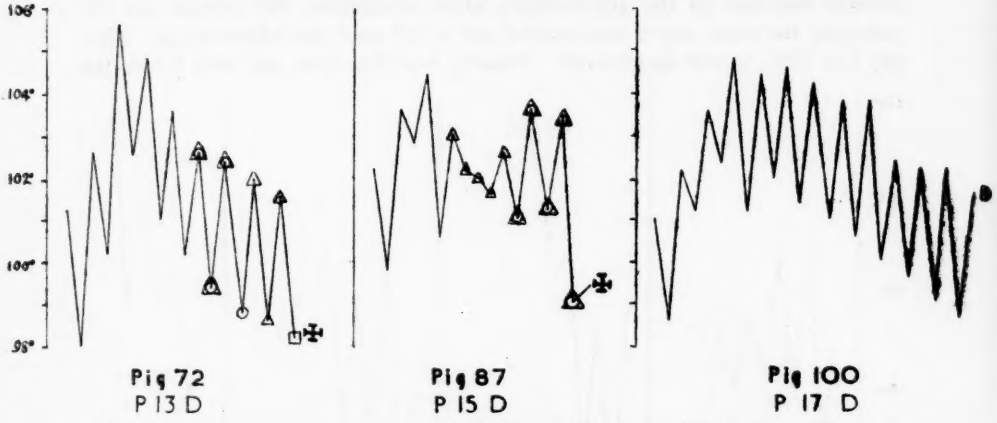

FIG. 6.—Temperature charts of pigs 72 and 87, which died with lesions suggestive of rinderpest.
The symbol Δ around a reading indicates *slightly off feed*; □ *complete anorrhexia*; and ○ *diarrhœa*.
Pig 100 was the pair to pig 99 (Fig. 4). It proved immune on challenge and illustrates the chart of an animal, late in the "direct" series, that was not killed for material.

of virus in P20D *bis* developed rinderpest by contact. The surviving pigs of passages P18D *bis*, P19D *bis* and P20D *bis* were immune on challenge with buffalo virus. The surviving pig of passage P21D *bis* reacted to pig-passaged goat virus (KP30).

At this time the development of the alternate line had reached a stage at which it appeared to be the more satisfactory of the two. There was little to be gained from continuing attempts to propagate the direct line, and experiments to this end were discontinued.

Alternate Passage in Pigs

The alternate line has been passaged 20 times in pigs, a pair of rabbits having been inoculated between each pair of pigs. The pigs have been inoculated intravenously with 5 c.c. of a 10^{-1} dilution of a suspension of 1 part

rabbit spleen and mesenteric gland to 20 parts of defibrinated blood. During the first 3 passages, rabbits were injected intravenously with 0.25 c.c. of a similar mixture prepared from pig spleen and blood; but later, 0.25 c.c. of a 10^{-1} suspension of pig-spleen in saline was used. With three exceptions, when they have been left 5 days, pigs have been killed for material on the 4th morning after inoculation.

The reactions in pigs during the earlier passages were comparable to those that occurred in pigs inoculated with the direct line. From the 15th or 16th passage, however, there has been a tendency towards a smoother type of curve (Fig. 8, pig 152), the morning temperatures describing an almost symmetrical arc over the course of 7 or 8 days. It seems probable that this more even type of curve is of greater significance than sudden rises to 104° F. or more. Pigs of passage P18A were relatively resistant—indeed, they were left until the 5th morning before one (pig 172, Fig. 7) was selected for slaughter. The rabbits inoculated from this pig reacted normally.

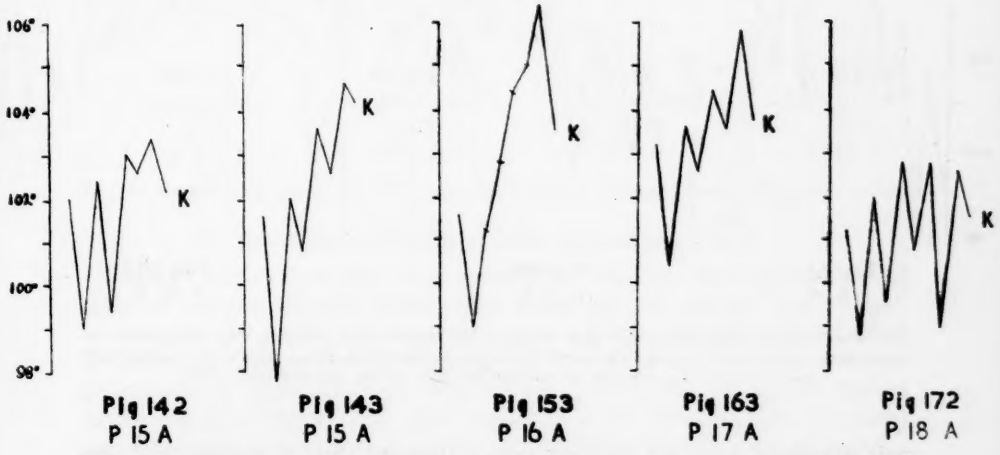

FIG. 7.—Temperature charts of some pigs of the "alternate" series for comparison with Figs. 4 and 5.

As with the direct line, one of each pair of pigs has been left for challenge with buffalo virus. Three of these pigs have died before inoculation with challenge virus. Pig 12 (P3A) died 27 days after inoculation, having shown inappetence for over 3 weeks. The fæces were abnormally soft from the 7th to 11th days after inoculation. This pig was noticed to be unsteady on its feet on the 3rd day, and later spent most of its time asleep. If roused, it was very aggressive. On post-mortem examination, no lesions of rinderpest were found. Pig 51 (P8A, Fig. 8) died on the 7th day after inoculation. It had suffered from diarrhœa and inappetence for 3 days, and mild lesions of rinderpest were found *post mortem*. On the morning of its death, it was found with its head

fast between the bamboo poles of which the walls of its sty were constructed and, no doubt, exhaustion from struggling was in part the cause of death. Pig 95 (P11A, Fig. 8) died during the 9th night after inoculation. There had been severe diarrhœa for 3 days and lesions of rinderpest were present.

In addition to these three pigs, three others of those left to survive developed diarrhœa for one or more days, and two more were temporarily off feed. All pigs which have been challenged with buffalo virus have proved immune.

In general, the rabbits inoculated between each pig passage have behaved like rabbits inoculated with fresh lapinised virus of rabbit origin. One of the

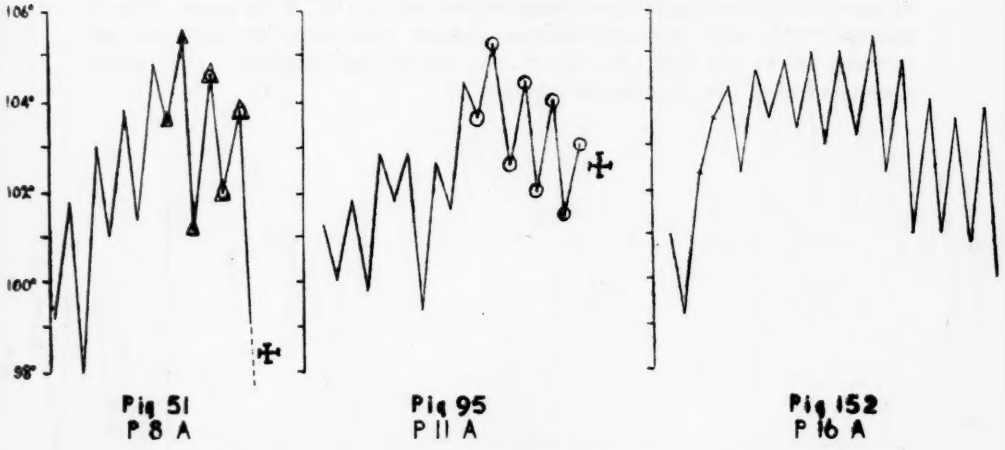

FIG. 8.—Temperature charts of pigs 51 and 95, which died with lesions suggestive of rinderpest. The chart of pig 152 shows the more symmetrical curve which, it is suggested, may indicate the reaction of susceptible pigs to the pig-passaged virus.

pair inoculated from the P3A pig gave a doubtful thermal reaction and was not killed. There have also been two deaths from coccidiosis, one before a reaction could be expected and one early in the reaction before lesions had had time to develop.

Rabbits inoculated with material of the first three pig passages (Fig. 9) tended to have the peak of the reaction about the 4th morning. Thereafter the incubation period seemed to decrease, and it was thought desirable to kill rabbits inoculated with the 5th, 6th, 7th and 8th passages during the 3rd day, usually after noon. From the 9th passage, the rabbits have been slaughtered on the 4th morning, the peak morning temperature usually having been on the 3rd day. Lesions have been exactly comparable to those found in rabbits inoculated with lapinised virus of rabbit origin: there has been the usual variation in the severity of necrosis at the different, recognised sites; but there has

never been doubt as to the presence of necrosis at one site at least. The fact, that the presence of virus at each passage is checked automatically by the reaction and lesions of the rabbits, is a most important feature of the alternate line. If a pair of very resistant pigs should be inoculated, the absence of virus in the spleen would be detected from the failure of the rabbits to respond, and further pigs could be inoculated with stored material with a minimum of delay.

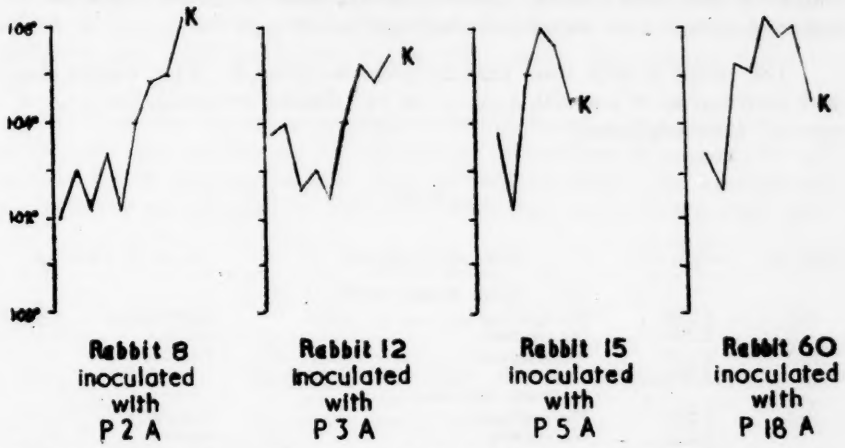
K
K
K
K
Rabbit 8 inoculated with P 2 A
Rabbit 12 inoculated with P 3 A
Rabbit 15 inoculated with P 5 A
Rabbit 60 inoculated with P 18 A

FIG. 9.—Temperature charts of rabbits inoculated with pig material in the "alternate" series.

Inoculation of Buffaloes with Pig-passaged Virus

The value of lapinised virus passaged in pigs for the immunisation of buffaloes was first tested with both lines at the 3rd pig passage. Pig 8 (P3D) was killed on the 4th day after inoculation and a portion of spleen was emulsified in defibrinated blood (1 : 20) following the procedure usually adopted in the preparation of lapinised virus from rabbits. From this suspension tenfold dilutions in physiological saline were prepared and two buffaloes were inoculated subcutaneously, each with 5 c.c. of the 10^{-1} dilution, two with 5 c.c. of the 10^{-2}, two with 5 c.c. of the 10^{-3}, and two with 5 c.c. of the 10^{-4}. One of the two that received the 10^{-2} dilution was off its feed on the 7th day after inoculation, showed dysentery on the 8th day, and died the following night. There had been no thermal reaction; but lesions found *post mortem* suggested that death was due to a natural infection with buffalo rinderpest. It should be recorded here that although the buffaloes under test were housed in an isolation shed about 60 yards from the infected shed and separated from it by a road, fenced on each side with barbed wire, large numbers of pariah dogs and jungle crows, which fed on the remains of carcases of animals killed for vaccine production, made strict isolation quite impossible. None of the other seven buffaloes showed any reaction or evidence of rinderpest. They were moved to the infected shed on the 14th day after inoculation and challenged by the inoculation of virulent buffalo-rinderpest gland and spleen suspension.

Similarly, with passage P3A, pig 13 was killed on the 4th day and a suspension of spleen in blood was made. Dilutions in physiological saline were prepared and eight buffaloes were inoculated. Three of these buffaloes were in poor condition and appeared to give thermal reactions. One, that received 5 c.c. of the 10^{-2} dilution, died of surra on the 4th day after inoculation: there were no lesions of rinderpest. Both the buffaloes that received the 10^{-3} dilution showed a suspicious thermal reaction but no other symptoms. The seven surviving animals were moved and challenged on the 14th day.

The results of both these tests are given in Table I. They suggest that with both batches of material, 5 c.c. of the 10^{-3} dilution protected; but 5 c.c. of the 10^{-4} were insufficient.

TABLE I

Dilution	*Buffalo No.*	*Result of Inoculation*	*Result of Challenge*
		P3D. FRESH SPLEEN : BLOOD : : 1 : 20	
10^{-1}	2.2329	No reaction	Immune
	2.2365	No reaction	Immune
10^{-2}	2.2323	No reaction	Immune
	2.2333	Died 8th night, probably of contact rinderpest	——
10^{-3}	2.2279	No reaction	Immune
	2.2321	No reaction	Immune
10^{-4}	2.2282	No reaction	Susceptible
	2.2308	No reaction	Susceptible
		P3A. FRESH SPLEEN : BLOOD : : 1 : 20	
10^{-1}	2.2480	No reaction	Immune
	2.2481	No reaction	Immune
10^{-2}	2.2384	Doubtful mild reaction	Died *surra* 4th day
	2.2482	High temperature, day of inoculation and next day only	Immune
10^{-3}	2.2483	Doubtful mild reaction	Immune
	2.2381	Doubtful mild reaction	Died 6th day, fluke; no lesions of rinderpest
10^{-4}	2.2484	No reaction	Susceptible
	2.2485	No reaction	Susceptible

In view of the many advantages to be gained from issuing a dried product for routine use, a further experiment was made with material from pig 13. Two c.c. samples of the suspension of spleen and blood were pipetted into 20 c.c. penicillin bottles. One gramme samples of minced spleen were also distributed in penicillin bottles, and all the bottles were attached to the manifold of a cryochem apparatus. The apparatus was run for $11\frac{1}{4}$ hours with a vacuum of 100 to 125μ of Hg. The material froze at once and drying appeared to be rapid. The original rubber cap, greased with soft paraffin, was then placed over the mouth of the bottle. A small hypodermic needle was inserted, attached to the pump and a good vacuum was obtained in each. The bottles were then placed at —20° C. Owing to shortage of buffaloes it was necessary to store

them for 23 days, and when they were removed from the storage cabinet it was found that there was no vacuum. However, 20 c.c. of saline were added to a bottle which had received 2 c.c. of spleen-blood suspension to give a dilution equivalent to 10^{-1} of fresh material. From this, further dilutions were prepared and 5 c.c. doses were inoculated to three pairs of buffaloes, one pair receiving the 10^{-1}, one pair the 10^{-2}, and one the 10^{-3} dilution.

To a bottle that had held 1 gramme of fresh spleen, 10 c.c. of saline were added to give a 10^{-1} suspension and further dilutions were prepared. Six buffaloes were again inoculated; but, in this instance, two received the 10^{-2}, two the 10^{-3}, and two the 10^{-4}.

The buffaloes showed no reaction to the inoculations. Unfortunately, one of the pair that received the 10^{-1} dilution of spleen-blood suspension died of barbone 8 days after inoculation. The results of challenging the survivors on the 14th day are recorded in Table II. From these results, it was clear that

TABLE II

Dilution	*Buffalo No.*	*Result of Inoculation*	*Result of Challenge*
		P3A. Dried spleen : blood : : 1 : 20	
10^{-1}	2.2842	No reaction	Doubtful reaction; died of barbone 13th day
	2.2843	No reaction, died of barbone 8th day	——
10^{-2}	2.2840	No reaction	Doubtful thermal reaction; no other symptoms; recovered
	2.2841	No reaction	Susceptible
10^{-3}	2.2838	No reaction	Susceptible
	2.2839	No reaction	Susceptible
		P3A. Dried spleen	
10^{-2}	2.2844	No reaction	Immune
	2.2845	No reaction	Immune
10^{-3}	2.2846	No reaction	Susceptible
	2.2847	No reaction	Susceptible
10^{-4}	2.2848	Doubtful mild reaction	Susceptible
	2.2837	Doubtful mild reaction	Susceptible

considerable loss of titre had occurred either during drying or during storage, and on theoretical grounds it seemed probable that loss of vacuum during storage might be to blame. Nevertheless, when a dose of 5 c.c. of a dilution equivalent to 10^{-2} of fresh spleen was administered, after drying and storage for 23 days under very indifferent conditions, spleen was capable of protecting two out of two animals.

In later titrations, minced spleen dried in bulk (about 40 g. in a 500 c.c. container) was used. Repeated tests showed that, when dried on the cryochem apparatus, pig spleen—like goat spleen—lost about four-fifths of its weight. In expressing dilutions, the figure given is always the equivalent of fresh material. Thus, in preparing a 10^{-1} dilution, 200 mg. of dried, powdered spleen (= 1 g.

fresh spleen) were emulsified in a Ten Broeck grinder in 10 c.c. of physiological saline. As the dose administered to each animal was 5 c.c., the actual weight of dried material given is expressed in grammes by the dilution figure. The buffaloes were challenged on or about the 14th day with buffalo-virus.

Table III gives the results of titrations with the direct line. P6D protected 2/2 buffaloes at 10^{-3}, 0/2 at 10^{-4}. P10D and P17D apparently protected 1/2 at 10^{-4}.

TABLE III

Dilution	*Buffalo No.*	*Result of Inoculation*	*Result of Challenge*
		P6D	
10^{-2}	2.2858	No reaction	Immune
	3.66	Died anthrax 12th day after inoculation	——
10^{-3}	3.02	Thermal reaction 11-15th days; recovered	Immune
	3.102	No reaction	Immune
10^{-4}	2.2857	No reaction	Susceptible
	3.105	No reaction	Susceptible
		P10D	
10^{-2}	3.139	No reaction	Immune
	3.140	No reaction	Very doubtful sharp reaction; recovered
10^{-3}	3.135	No reaction	Immune
	3.137	No reaction	Immune
10^{-4}	3.130	No reaction	Susceptible
	3.131	No reaction	Immune
		P17D	
10^{-2}	3.250	No reaction	Immune
	3.253	No reaction	Immune
10^{-3}	3.247	No reaction	Immune
	3.251	No reaction	Immune
10^{-4}	3.215	No reaction	Immune
	3.256	No reaction	Susceptible

Table IV, which shows the titrations with the alternate line, requires more extensive comment. Six buffaloes were inoculated with P6A; but four are omitted from the table as they contracted rinderpest by contact. It would seem, however, that this batch failed to protect at 10^{-3}. The first titration of P10A was unsatisfactory. One of the buffaloes that received the largest dose was not protected. The other, and both those receiving the next dilution, gave doubtful thermal reactions on challenge and recovered. This suggested that the first buffalo was so resistant that it failed to respond to the inoculation of the mild lapinised virus, but proved susceptible on challenge with the stronger buffalo-virus. Examples of such behaviour will be given later. From the number of buffaloes, from the same source, that failed to give satisfactory reactions or die, when inoculated with buffalo-virus for the production of

inactivated vaccine, the odds of including three immune buffaloes in a group of six taken for a titration experiment were found to exceed 1 : 6,000. The possibility that buffalo 3.159 was somewhat resistant to rinderpest was enhanced by its behaviour to the challenge dose. It reacted late, and the temperature rose to a greater height than is usual with fully susceptible buffaloes. Although

TABLE IV

Dilution	*Buffalo No.*	*Result of Inoculation*	*Result of Challenge*
		P6A	
10^{-3}	2.2852	No reaction	Susceptible
10^{-4}	2.2855	No reaction	Susceptible
		P10A	
10^{-2}	3.159	No reaction	Susceptible
	3.161	No reaction	Very doubtful thermal reaction; recovered
10^{-3}	3.162	No reaction	Very doubtful thermal reaction; recovered
	3.163	No reaction	Very doubtful thermal reaction; recovered
10^{-4}	3.164	Temperature high day of inoculation and following day only	Susceptible
	3.165	Doubtful mild reaction	Susceptible
		P10A—RETEST AFTER 35 DAYS AT —20° C.	
10^{-2}	3.314	No reaction	Susceptible
	3.337	No reaction	Susceptible
10^{-3}	3.309	No reaction	Susceptible
10^{-4}	3.275	No reaction	Susceptible
	3.317	No reaction	Susceptible
		P15A	
10^{-2}	3.346	No reaction	Possible mild reaction; recovered
	3.347	No reaction	Immune
10^{-3}	3.338	No reaction	Immune
	3.351	No reaction	Immune
10^{-4}	3.344	No reaction	Susceptible
	3.348	No reaction	Susceptible
		P19A	
10^{-2}	3.399	No reaction	Immune
	3.403	No reaction	Immune
10^{-3}	3.400	No reaction	Immune
	3.404	No reaction	Immune
10^{-4}	3.407	No reaction	Susceptible
	3.412	No reaction	Susceptible

dysentery developed later in the day, the fæces were only slightly soft on the 9th morning early. However, on repeating the titration, with dried material stored for 35 days at —20° C., it was found that the batch failed to protect at a dilution of 10^{-2} and it is clear that it was of low virus content.

P15A and P19A had a titre almost as good as that of P10D and P17D, and for this reason there seemed no point in continuing the direct line. An experiment was, however, made to find out if three direct pig-passages after seventeen

alternate (P17A3D) would result in material with a higher titre. This material was dried by two procedures and both dried products were titrated. In each case the buffaloes were protected by the 10^{-3} dilution. It would appear, therefore, that the three direct passages did not improve the titre.

Drying and Storage Experiments

In most of the experiments dried virus was prepared by distributing minced spleen in bottles, fixing to the manifold of a Model 102 cryochem apparatus (F. J. Stokes Machine Co.), and running the vacuum pump until the evening. With a vacuum of 250 to 100μ of Hg, freezing occurred rapidly and no attempt to cool the containers was made. The apparently-dry material was stored *in vacuo* at —20° C. overnight. In the morning, it was broken up with a glass rod and the bottle was replaced on the apparatus. The pump was run for a further 10 to 12 hours, after which the material was ground to a powder in a mortar and two or three samples of 200 mg. were weighed into glass tubes which were sealed under vacuum. The balance was stored in bulk. During the early work, practical difficulties prohibited final desiccation in individual containers and sealing "off the manifold." In the case of P15A, however, some material was weighed into final containers, dried on the third day for a further period of 5 hours, and sealed before removal from the manifold.

By the time that the P19A and P17A3D pigs were killed, an Edward's centrifugal freeze-dryer had been installed. With these batches spleen was dried, on the first day, in bulk on the cryochem apparatus. With P19A and one portion of P17A3D, after powdering, a second day's drying on this apparatus was given. The other portion of P17A3D was placed in a beaker in the centrifuge chamber of the primary system of the Edward's machine and dried with heat (37° C.) for the same period. On the third day, all three lots were weighed in 200 mg. amounts into glass phials and dried for 8 hours on the secondary system of the Edward's machine before sealing.

There was little difference in titre between P17D, P19A and the two lots of P17A3D. It would seem, therefore, that neither exposure to room temperature (about 30° C., occasionally above) for a third day, nor the extra 7° C. to which it was exposed when the heater of the Edward's primary chamber was used, had any markedly adverse effect on the titre. Although no comparative titrations of fresh material of later passages were made, it seems clear that the pig spleen can be dried without serious loss of titre. From this aspect the preparation of dried virus for issue is, therefore, practicable.

As to the keeping properties of the dried product, the tests that have been made can only be regarded as preliminary. A trial was made with P10D. This material was dried for 11½ hours on the first day and 7 hours only on the second, a total period of 18½ hours. It was then weighed out in 200 mg. amounts into glass ampoules and, without further drying, sealed *in vacuo*. One

tube was titrated after 6 days storage at —20° C. Two tubes were, at the same time, removed from cold storage and left in a closed box on the bench at a temperature of 27 to 30° C. After a week, one of these was titrated and, after a fortnight, the other. A fourth tube was sent to Bangkok, then by post by Chiengrai, back to Bangkok and then to the laboratory. It was away from the laboratory 14 days, after which it was titrated.

The material, not exposed to room temperature, protected both buffaloes at 10^{-3}, and one of the two that received the 10^{-4} dilution proved immune at challenge (Table III). After a week at room temperature, both the buffaloes that received the 10^{-2} dilution resisted challenge; but those receiving the 10^{-3} dilution proved susceptible. After a fortnight only one of the pair inoculated with the 10^{-2} dilution was immune. All the buffaloes inoculated with material sent to Chiengrai (lowest test dilution again 10^{-2}) proved susceptible.

A batch of the 10th passage of Kabete attenuated goat (KAG) virus in pigs (KP10) dried similarly, but only for a total of 15 hours, was tested simultaneously. When titrated soon after drying, this virus protected 2/2 cattle at 10^{-4} and 1/2 at 10^{-5}. After exposure for a week to room temperature (in the same box as the P10D), it protected buffaloes at 10^{-4}. After exposure for a fortnight at room temperature, it still provoked a reaction followed by immunity in both buffaloes that received the 10^{-4} dilution. Material sent to Chiengrai provoked a reaction and immunity in 1/2 buffaloes at the same dilution: the other did not react and was susceptible on challenge.

This experiment showed that there was a marked difference between the keeping properties of lapinised and goat viruses when prepared in pigs under similar conditions. Whilst the result may be due to a difference in attacking power between the two strains rather than to a difference in quantity of virus, it suggests that the former virus requires a better drying technique before it can be expected to withstand a reasonable exposure to atmospheric temperatures. Further experiments with material dried for a longer period, on the third day, in individual containers which can be sealed whilst still attached to the manifold, are being made. From the experiments recorded above it is known that this extra drying will do little harm to the virus titre.

Field Experiments

Before any attenuated virus can be used for immunisation in the field, it is necessary to determine whether it has sufficient attacking power to provoke response in a reasonable percentage of animals of the type on which it is likely to be used and the minimum dose required. Buffaloes for all the experiments recorded in this paper were obtained from the southern part of Thailand, from an area which has been free from rinderpest for many years. Although the percentage which, when inoculated with virulent buffalo-virus, failed to react sufficiently well to be suitable for vaccine production was below five, susceptibility could not be considered absolutely uniform. In particular, the type of

thermal response varied. The most susceptible animals appeared to offer no resistance at all. Dysentery developed about the sixth day without, in many cases, an increase in temperature of more than 1° F. As the resistance of the buffaloes increased, there was a more marked thermal response and a delay in the onset of other clinical symptoms. An identical response by all buffaloes to a very mild virus was, therefore, not to be expected.

An experiment was made with batch P15A: the method of drying and laboratory titration have been described above.

A batch of buffaloes was purchased and railed to Muak Lek, a veterinary quarantine station on the railway about one hour's journey from the laboratory. The dried virus was taken to Muak Lek in a vacuum flask containing ice, and exposure to temperatures above freezing point was minimal. No attempt was, however, made to cool the saline or mortar and pestle used in preparing the suspension, and the conditions generally were such as could be attained without difficulty at any centre which could be reached before the ice in a large flask had melted. Eighteen buffaloes were inoculated with a suspension of dried virus so that each received the equivalent of 5 mg. in 5 c.c. of saline. A fortnight later two of these buffaloes were walked to the laboratory and challenged. Both proved immune, and a further fourteen were then sent up. Of these, ten proved immune and four susceptible. Under the conditions of the experiment, therefore, a 5 mg. dose protected twelve out of sixteen buffaloes, a rate of 75 per cent.

Later a further thirty-six of the buffaloes at Muak Lek were inoculated with doses equivalent to 10 mg. of dried virus in 5 c.c. of saline. Fourteen days later thirty-three were sent for challenge, and all proved immune. Control buffaloes from the batch at Muak Lek were included in each lot forwarded to the laboratory and all proved susceptible.

As was expected, dosage was proved to be of great importance. Five mg. did not protect all the animals, but by increasing the dose an excellent cover was obtained. There is no doubt that the dose of a mild virus which will elicit a response in some animals may fail to provoke immunity in others of slightly higher resistance and, in determining a suitable dosage for use in field inoculations, a large margin over the minimum infective dose—as determined in a laboratory titration experiment—must be given. In this instance 1 mg. of dried virus protected 2/2 buffaloes in the titration; but five times this dose was not sufficient to give a satisfactory cover in the field.

It may be mentioned that none of the buffaloes inoculated at Muak Lek showed any clinical symptoms. Failure to obtain all the inoculated buffaloes for challenge was due to accidental causes: for example, horn wounds made some unfit to walk to the laboratory.

Although the inoculation of buffaloes in the field with dried pig spleen proved so satisfactory, a small experiment on its value for immunising the

highly resistant local cattle showed that it was quite unsuited to this purpose. Seven cattle only were challenged, a fortnight after inoculation with 5 mg. of dried spleen, and all reacted. It would appear that the animals were too resistant to respond to the mild lapinised virus. However, the less attenuated goat-virus has been used with great success for the protection of cattle in Thailand.

Discussion.

In view of the lack of resistance to rinderpest of the water-buffaloes in Thailand it is necessary to employ for their immunisation either an inactivated vaccine or a virus of relatively low virulence. Fresh lapinised virus was found to be of suitable virulence for field use; but the small supply of rabbits limited its employment. By utilising pigs, ample supplies of fresh material can be obtained without difficulty. Passage in pigs does not enhance the virulence of the strain for buffaloes.

Although the virus appears to be much more delicate than KAG virus, drying experiments show that infected pig spleen can be dried without difficulty to give a product of reasonable titre. Further work is necessary to decide on the drying procedure that will give a preparation with optimum keeping properties. Storage at low temperatures presents little difficulty; but, for use under field conditions, the product should be able to withstand a few days' exposure to atmospheric temperature without a heavy drop in titre.*

Compared with inactivated vaccine, the lapinised virus in the form of pig spleen is much cheaper to prepare. Pigs of the size used in the experiments have spleens weighing about 60 g. and yielding about 12 g. of dry powder. At a 10 mg. dose about 1,200 doses are obtainable from one pig, whereas one buffalo gives about 200 doses of inactivated vaccine.

Unfortunately, no information could be obtained in the time available on the relative duration of immunity induced by the two products. Since inoculation of lapinised virus into buffaloes provokes no thermal response, it appears unlikely that a quasi-permanent immunity, similar to that induced by KAG virus in cattle, will result; and in countries which are constantly threatened with invasion by rinderpest, the advantages of a long-term immunity are too obvious to need comment. The ideal attenuated virus for immunisation is probably one that will cause a mild temperature reaction without other clinical

* Since this was written, some information on the keeping properties of P19A has become available. A tube of dried virus was brought from the laboratory to Bangkok on ice and was there stored at +4° C. for three nights. It was then brought by air to England in a jacket pocket, being exposed to varying atmospheric temperatures during 72 hours. On arrival, it was placed at —20° C. and stored at that temperature for about a month. The tube was then taken to Egypt by air, again travelling without refrigeration. The authors are grateful to Mr. R. Daubney, C.M.G., O.B.E., who kindly tested the virus on rabbits and on Egyptian cattle.

In rabbits the virus provoked typical reactions and lesions.

Twenty-four cattle were inoculated, each with a dose of 10 mg. of dry material. Of these, 11 reacted and were immunised: 6 failed to react but were immune on test: 5 failed to react and were susceptible on challenge: 2 reacted to both the original and challenge inoculations. Of 20 controls, 19 reacted typically to the challenge dose. It appeared that the virus was still capable of protecting about 75 per cent, a percentage similar to that protected by straight lapinised virus in this type of animal. J.R.H.

symptoms. Future work on virus attenuated in the chick-embryo or by other means may lead to the development of such a strain. It may be mentioned that KAG virus passaged in pigs has fallen in virulence for buffaloes and it is possible that, with further passages, a strain suitable for the field immunisation of these animals may result. In the meantime pig-passaged, lapinised virus may be of some service.

Summary

A strain of Nakamura III lapinised virus was passaged successfully both directly from pig to pig for seventeen passages and alternately in pigs and rabbits. For the production of virus for field use, the latter procedure is the more satisfactory.

Local Thai pigs vary greatly in susceptibility to rinderpest and it is, therefore, preferable to use dried, tested virus prepared in the laboratory rather than fresh virus of uncertain titre prepared in the field. Although delicate, the virus can be dried without undue loss of titre. Further work is necessary to determine the best procedure to give a product with reasonable keeping properties at atmospheric temperatures.

The virus provokes no clinically-detectable response in water-buffaloes in Thailand. When transported on ice and exposed to atmospheric temperatures for a minimum period, 10 mg. of dried powder produced immunity in every buffalo of a group inoculated as a field experiment. The virus was of no value for the protection of the relatively-resistant local cattle.

No tests were made on the duration of immunity in buffaloes. In view of the fact that there is no thermal response to the inoculation, this may not be great.

Acknowledgments

The authors are indebted to Khun Vichitr Pahanakarn, Nai Tiang San and Nai Lek Jayavasu, without whose help these experiments could not have been made; to Nai Archit Chôtisen, Nai Seri Suphankasen and Nai Samran Varn Briksha, for assistance in the work; and to Mr. R. P. Jones, for the suggestion that pigs might prove a suitable experimental animal and for his continual advice and encouragement.

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Skull of badger, showing separated portion of sagittal crest.

A UNIQUE BADGER'S SKULL

By GUY L. ACKERS,

Banstead.

THE carcass of a badger was found in a ditch at Gratton Park, near Merstham. The abdomen had been severely injured by gunshot. Whilst the skull was being cleaned it was noticed that there was evidence of multiple wounds, incurred on some previous occasion, and from which the animal had recovered. A portion of bone, 4.2 cm. in length, had been broken off the sagittal crest. Three calluses had been produced, one 9 mm., one 5 mm. and the third 1 mm. in length. The separated piece of bone had been displaced forwards, leaving a gap of 3 mm. between it and the parietal bone. There was a partly ossified hole in the vomer 3.5 mm. in diameter and a lead pellet, the obvious cause of the hole, was lodged in the right orbital cavity exactly halfway between the post-orbital wall and the lachrymal foramen. It had not caused injury to eyeball. Three more pellets were found lodged in the left maxilla, partially embedded in callus. Another hole similar to that mentioned previously was found in the mandible below the left lower canine. Both lower canines were badly broken, and also the three left lower incisors were in the same state.

It is presumed that these injuries were the result of gunshot.

The object of this note is to demonstrate that this badger had suffered from gunshot injury on two occasions, and that although the first injury was to a vital part, i.e., the head, the animal had been able to make a good recovery.

[EDITOR.—No doubt this poor beast went through a period of great suffering before it was restored to reasonable health, and this induces the editor to plead that gunshot wounds in wild animals, although they may be occasioned in vital parts, should not be presumed to be immediately fatal, and one should take all necessary steps to put such injured creatures out of their misery.]

REVIEWS

MY BEST ANIMAL STORY. London: Faber & Faber. Price 8s. 6d.

THERE are few who do not find delight in a well-told story in which some animal plays the major rôle. It is immaterial if the principal actor be portrayed in the guise of a noble hero, a cunning, vicious knave, or a humble, timid, self-effacing creature. We love them all.

There are authors who specialise in this type of literature and are so gifted in their art that one follows their story not from a ringside seat, but as a complete participant in the triumphs and tribulations of the actors.

This book is a collection of twenty-four such stories, each by a famous author, and each author has chosen the best from his list.

It would be invidious for one reader to assess them in order of merit; suffice it to say that they are all good, and the reader can look to many hours of delightful entertainment. It may be selfish advice, but I would warn a prospec-

tive purchaser to keep his book to himself until he has read it, for if it should come to the knowledge of the family circle, competition will be so great that his enjoyment may be postponed indefinitely.

DOLLAR'S VETERINARY SURGERY, by J. J. O'Connor, M.R.C.V.S. Pp. xiv + 1,036, with 403 Figs. Fourth edition. London: Ballière, Tindall & Cox. Price 35s.

"DOLLAR'S Veterinary Surgery" was first published at a time when the horse was the animal of prime interest to veterinary surgeons, and it immediately attained the rank of a classic. It has held that position ever since, and, indeed, there is no book which can compare, or even compete, with it as an all-round reference to all matters appertaining to the art and science of veterinary surgery.

Since Prof. O'Connor undertook the task of editing or revising the book, we have noted many alterations from the original text, many more references to surgical conditions and techniques in the other domestic animals, and also the inclusion of new material as the occasion might necessitate. In this edition the antibiotics, the sulpha drugs, new methods for inducing a state of anæsthesia, modern methods of bone repair and of dehorning cattle are introduced.

At the end of the book he has included in Appendix i and ii most valuable information on the examination of horses as to soundness and on identification —I am glad that Professor O'Connor has not departed from the original main theme of the book, which was to take the surgical diseases of the horse, as it were, a type, and to deal by comparison with those of the other domestic animals. Surgery or medicine is basically the same, no matter what animal, be it even man, comes under review. It is a well-recognised fact as regards veterinary science that those who become proficient in the equine field can turn their attention and with competence to any other species if the occasion should arise. The same state of affairs does not exist in regard to those who, for example, turn their attention to the ox or the dog. Now, the average modern graduate comes into the latter category, and his knowledge of the horse, its diseases, and the therapeutics which can be applied is little short of lamentable. As regards his ability to apply methods of restraint for operative purposes, the less said the better.

This book can therefore serve a better purpose than the first edition. In 1912, much of the information contained in its pages was common knowledge amongst veterinary surgeons. If the young people of to-day will only read and digest its contents they will profit much.

Professor O'Connor has deleted some illustrations and added others, but I do regret that he has dispensed with those fine old pictures depicting methods of casting and securing horses for operation. These pictures would serve a more useful purpose to-day than they did in 1912. Any surgical consultant can prove this to be true. In 1912, and even as late as 1930, he could rely on the veterinary surgeon in charge of the case to prepare the patient for operation, to cast, anæsthetise and secure. The consultant could confine his attention to the operation with no extraneous anxiety. Nowadays this is not the case, for on most

occasions the consultant has to step in and do all these jobs if he wants to avoid a catastrophe.

There are some "modern" surgeons who openly criticise this book. To them I will quote a cryptic remark of Professor Shave, given on a similar occasion: "It's because you don't know."

I welcome this new edition of a great book which is being so ably kept "evergreen" by Professor O'Connor.

I do wish that the publishers would make it of such a shape that it would lie easier on the hand.

MORLEY'S DAIRY FARMERS' ENCYCLOPEDIA, by Alan Morley, with foreword by John Hammond, F.R.S. Andrew George Elliot, Glade House, Kingswood, Surrey. Price 5s.

A MODERN dairy cow is a milk-producing machine, and in many ways she may be likened to the wonderful mechanical contraptions which are used in industry to-day. Both have been developed under the urge of economic demand, both are the result of man's ingenuity, and both are far removed from their relatively simple precursors.

The factors which have enabled the modern dairy cow to evolve may be summarised as follows: Environment, selective breeding, nutrition, and management, and the farmer has been compelled to improve under the spur of economics.

Until quite recent times, and although much had been achieved, efforts to improve were, in the main, empirical and the result of a system of trial and error.

Such a system should not be condemned, for it has given good results, and our modern animals of all species are the consequence of such efforts on the part of our forebears. Improvements which come about in this way are slow to mature, and they may be somewhat haphazard. However, they do give the scientist an opportunity to work back for an explanation, and when once that has been made clear the forward march can be accelerated and with more precision. Since our scientists have turned their attention to the dairy cow, great progress has been made, and the economic results are so obvious that breeders and farmers, in many ways the most conservative of men, have not been slow to take advantage of the new knowledge.

Much of this knowledge is stored in books and scientific papers far beyond the ordinary man's comprehension. Unfortunately, scientists are apt to be abstruse, and there is a great need to enterpret their findings in such a way that other people can understand them and apply them in the field.

This small book goes far to fulfil that requirement as regards the dairy cow. It is written in a simple style and language that any ordinary, intelligent person can follow.

In fourteen short chapters the author brings under review the basic anatomical and physiological facts, the various breeds, how to build up, feed and manage a dairy herd. There is also a chapter on beef cattle. Milking and clean milk production and health and disease are also discussed. At the end

of his dissertations, four pages are devoted to a summary which makes interesting reading.

There are three appendices on "Books for Further Reading," statistical tables of the "Nutritional Value of Feeding Stuffs," and on "Grades of Milk."

The book can be recommended to all those who are interested in the improvement of our dairy herds.

AN ATLAS OF CAT ANATOMY, by H. E. Field and M. E. Taylor. London: Cambridge University Press. Price 28s.

THE anatomy of the cat receives scant attention in most veterinary schools. There are several reasons why this should be so: a curriculum already overcrowded, the general similarity in structure of the cat and the dog, and the persistence of the prejudice that the cat is hardly worth bothering about. It is true that there is little time to spare for detailed consideration of the cat, but, as this animal now occupies much of the time of many practitioners, no veterinary student or practitioner can afford to be entirely ignorant of its more striking anatomical peculiarities. He should at least know enough to avoid such gross errors as the common one of interpreting the clavicle as a foriegn body when seen in radiographs.

This book has been prepared as a dissection guide for biology students and takes the form of a series of 57 plates, with a concise description of each on the facing page. Some of the plates are photographs, some drawings: most are good, though some of the parts illustrated by photograph would have been clearer if presented in diagrammatic form. For example, the photographs of the vessels and nerves of the neck, plates 51 and 52, are confusing: the diagrams of the thoracic vessels and nerves, e.g., plate 47, are much clearer.

Intended for a biology class, only the more important structures are labelled and described. Most veterinary users will not find this a disadvantage. Physiologists and others requiring a reference work on cat anatomy will be disappointed in the absence of detail, especially of the nervous system: it would not be just, however, to criticise this atlas on this score, as it seems to be very satisfactory for its declared purpose.

There is a short list of general references, an extensive glossary and an index. The book is produced in the new common American style with spiral metal binding: the general production is good, but no more. The price is no doubt reasonable in these times, but will deter most veterinary students from purchase: they would do well, however, to borrow the book and make at least a brief study of the plates.

DIE BIOCHEMISCHEN GRUNDLAGEN DER TIERNAHRUNGSLEHRE, by Professor Karl Scharrer. Stuttgart: Ferdinand Enke, 1950.

IN this paper-backed well-printed volume of just over 150 pages, the author gives an outline of the biochemical bases of animal nutrition. The first two-thirds or more of the book are devoted to an account of the chemical properties and metabolism of carbohydrates, fats and other constituents of living tissues,

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the amount of space devoted to each section being fairly proportional to its importance. The section dealing with vitamins, however, appears neither to be exhaustive nor up to date.

The final portion of the book deals with such matters as the total and digestible components of natural foodstuffs, balance of matter and of energy, and the nutritive value of foods, the last named being, perhaps, the best section of all.

In so severe a compression of material as this, choice of subject matter is important. The reviewer can see little reason for the inclusion in a book on animal nutrition of a section dealing with synthetic fats derived from hydrocarbons, regardless of its being will written. Moreover, of the references, three-quarters are to German work, indicating either a bias in selection or, as actually seems to be the case, that the book is intended mainly for German consumption. In the eleven pages devoted to the nutritive values of foods, Armsby's work in America and the standards proposed by the Scandinavian workers Hansson and Möllgaard are dismissed in about a page and a half.

As the title indicates, the scope of the book is limited to the biochemistry of nutrition. So far as veterinary students are concerned, they already have to cover a wider field of biochemistry than this. Furthermore, their syllabus in animal nutrition extends to a knowledge of the common foodstuffs and of the feeding of the different species of domestic animals, neither of which subjects is dealt with in the book. Its value to English students is, therefore, rather doubtful. With regard to post-graduate workers, the volume will be mainly of use as a source of references to German work in the biochemistry of nutrition.

N.A.A.S. Quarterly Review:—The Journal of the National Agricultural Advisory Service. No. 9. Autumn, 1950.

This review is published quarterly by His Majesty's Stationery Office for the Ministry of Agriculture and Fisheries. Price One Shilling.

This journal is essentially an abstracting one; the current issue contains some 18 abstracts of annotations concerning subjects such as, crop husbandry, virology, insecticides and fungicides, farm machinery, soils, which are of interest mainly to the agriculturist and/or horticulturist.

The veterinary surgeon, however, will find abstract under the following headings to have a direct bearing on his work:—animal breeding, poultry husbandry, and dairy husbandry. Under the last named, one finds annotations dealing with recently published work on milking technique, rate of milk flow, effect of mastitis infection on yield and chemical quality of milk.

Under "Animal Breeding" there are two abstracts dealing with "The Relation of Pedigree Promise to Performance of Proved Holstein—Friesian Bulls" and "Welsh Mountain Ewes as a source of Grassland Breeding Ewes."

The Poultry Husbandry section in this issue comprises two annotations, viz: "Turkey Production," and "Poultry Feeding."

Reading over the list of abstractors, one gains the impression that they are all skilled in their particular subject.

E.C.O.

OBITUARY

Emeritus Professor John Share Jones

WHILST this issue was still in the hands of the printers the sad news of the death of Professor Share Jones became known. He died on December 2, 1950.

Professor Share Jones was a man who gave a life of devotion to his profession. For fifty years he played a leading rôle as teacher, administrator and legislator. His interests and influence extended far beyond the confines of ordinary professional matters, for he took an active part in local and national affairs.

His passing leaves a gap in our ranks which it will not be easy to fill. We will all miss this virile, happy man, who was gifted with great vision and had all the attributes of a leader.

We intend to publish in a subsequent issue a tribute to his life and work.

To Dr. Mary Share Jones, his devoted wife, we extend, on behalf of our readers, the publishers and the editors of this journal, this immediate expression of our deepest sympathy in her great loss.

Publishers' Notices

THE BRITISH VETERINARY JOURNAL, with which is incorporated THE VETERINARY JOURNAL, is published monthly, and copy for advertisements should be in the hands of the publishers—Baillière, Tindall and Cox—not later than the 20th of the preceding month, if proof is required. Tel.: Temple Bar 3386.

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